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# Viral Causes of Respiratory Diseases in Swine

by

Leslie Hemmingson\*

## INTRODUCTION

Respiratory disease is one of the most frequently encountered pathological conditions in swine. The history of respiratory conditions in swine is indeed mystifying. At one time it was presumed that the majority of chronic pneumonias were associated with a virus causing enzootic pneumonia or with lung worms. It now seems probable that there are many primary causal agents.

Lamont (1952)<sup>5</sup> described a pneumonia caused by an influenza type virus and one caused by a filter-passing agent described by Gulrajani and Beveridge (1951). Betts (1952)<sup>1</sup> termed the disease caused by the filter-passing agent "virus pneumonia of pigs" (VPP). In 1956 it was shown that a strain of VPP virus would produce both typical and constant histopathological changes when the pneumonia was experimentally induced, but the examination of material from field cases showed a wide microscopic variation. From the reports of pathologists it appears that VPP, or a very similar form of chronic pneumonia, is widespread in France, Holland, Norway, Finland, Poland, Australia, South America, Canada, and the United States.

Lamont (1952)<sup>5</sup> states that virus pneumonia in pigs is a condition very similar to "Ferkel grippe" as described by Kobe and Woldmann (1933, 1934, and 1935). The disease is also similar to that described by Dorset (1922) and Shope (1931).

Swine influenza was apparently first

recognized in America as a clinical entity in the autumn of 1918. Koen, according to Shope (1931) called this new disease of pigs "flu" because of the similarity in symptoms to those shown by humans being affected by the pandemic of influenza which was then raging.<sup>10</sup> In 1928 Shope found many typical cases of swine influenza in eastern Iowa, but in 1929 much difficulty was encountered. A loose diagnosis of "hog flu" was made in many cases. These animals, as a rule were suffering from a respiratory condition quite similar to true epizootic influenza in some respects but differing markedly in others. The condition was usually recognized by the absence of prostration and by the greater chronicity of the disease as well as by the failure of more than a small portion of the herd to become infected.

Young and Underdahl (1955)<sup>14</sup> found that the influenza viruses appeared to cause only a part of the respiratory disease in swine. Assuming that the hemagglutination-inhibition titers and the serum neutralization tests were indicative of the incidence of swine influenza, they found that between 15 and 35 per cent of the swine in the United States are affected. This is a considerably lower incidence of respiratory disease than the lung lesions in swine coming to slaughter in the average packing plant would indicate, as 50 to 70 per cent of such lungs show evidence of some current or past respiratory disease. Since the influenza viruses appear to cause only a part of the respiratory disease among swine, Young and Underdahl considered other diseases which may be responsible for the lung lesions observed

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in slaughtered swine. In the last few years a disease or diseases which show gross pathological changes of the lungs indistinguishable from swine influenza has been reported from many different countries. This disease has become known as virus pneumonia of pigs.

There has been a great deal of confusion about the etiology of respiratory disease in swine. It is now, however widely accepted that there are two main etiological factors involved in viral respiratory disease in swine. The two, now distinct disease entities, are swine influenza and virus pneumonia of pigs.

### **ETIOLOGICAL AGENTS AND PREDISPOSING CAUSES**

Swine influenza is a disease of complex etiology being caused by infection with the bacterium *Hemophilus suis* and the swine influenza virus acting together. *Hemophilus suis* is a small Grams—negative, non-motile bacterium that shows a marked tendency towards pleomorphism. *Hemophilus suis* is not pathogenic for swine when administered alone in pure culture by way of the respiratory tract.

The swine influenza virus is pathogenic for white mice and ferrets as well as for swine. When administered alone to swine by way of the respiratory tract it causes a transient, mild illness that is clinically distinct from swine influenza.<sup>11</sup> Swine influenza virus is identical in size with type A human influenza virus and is very closely related antigenically.

Although neither *Hemophilus suis* nor the swine influenza virus alone produces a serious pathological condition, the two, when administered together by way of the respiratory tract cause a clinical illness identical with swine influenza seen in the field.<sup>12</sup>

Kobe (1934), according to Lamont, claimed that the virus of swine influenza persists for a period of time in the lungs and that the disease may be transmitted by mixing healthy pigs with survivors of an outbreak.<sup>5</sup>

The infective agent of virus pneumonia of pigs is a large virus probably not less than 200 millimicrons in diameter. It

has been shown to be susceptible to Aureomycin to some degree. The virus persists for long periods of time in the pneumonic lung, therefore the lesions rarely undergo resolution. Betts (1952)<sup>1</sup> recovered the virus from a pig known to have been infected 66 weeks previously and from a group of pigs known to have been infected 21 weeks previously. The long persistence of the virus in the lungs of infected swine results in continuous enzootic infection within the affected herds.

The natural host for virus pneumonia of pigs is the pig. No other natural or experimental host has been found.

Gordon (1963)<sup>3</sup> reported that the examination of 1,000 pig's lungs from each of two sources showed that both the incidence and the degree of pneumonia was lower in the pigs kept in an environment of high humidity and temperature. These pigs were raised under a condition of an average temperature of 80 degrees and a relative humidity of 95 per cent. The incidence of infection by VPP is directly related to the incidence of infected dams, the opportunity for distributing infection among suckling litters, the degree of mixing among weaned stock, the extent to which continuity of stocking is maintained in buildings, the density of stock per unit area, the quality of housing (referring particularly to methods of ventilation), the season of the year, and the type of nutrition. Although the virus alone may set up the lesions, in most natural cases, secondary bacteria are also present.

### **CLINICAL SIGNS AND SYMPTOMOLOGY**

Swine influenza is a herd illness, not a disease of the individual. It is essentially a disease of early fall and winter. Ordinarily, within a period of 24 hours after the first signs of the disease, the majority of a herd will show clinical symptoms. The disease has a sudden onset with sneezing and coughing as prominent symptoms; however, a temperature rise to at least 104 degrees Fahrenheit is the first absolute evidence of illness. Accompanying

the temperature rise, or shortly thereafter, is a mild degree of malaise, mild anorexia and a tendency for the animal to become easily fatigued. On the second and third day of illness the temperature rises and anorexia and malaise are more severe. "Thumping" respirations are usually in evidence on the third or fourth day. The condition of the fourth and fifth days is little altered from that on the third day. Death may occur on any day after the first or second and is usually preceded by an exaggeration of respiratory symptoms, increased prostration, and an incoordinated delirium. Fatal cases usually exhibit extreme edematous broncho-pneumonia as the cause of death.<sup>5</sup> Generally, the temperature of the survivors decreases and recovery is uneventful and rapid.

According to Shope (1931)<sup>9</sup> swine influenza has a morbidity of near 110 per cent and a mortality of one to four per cent. There is usually some stressful condition preceding an outbreak of influenza in a herd of swine. This stress may be due to cold, wet weather or merely to a change in feed or some other management condition.

Virus pneumonia of pigs may take an acute form or, more frequently, a chronic form. Typically, pigs begin to show symptoms at three to ten weeks of age. The condition is first noticed as a transient diarrhea followed in a day or two by the development of a dry non-productive cough. The cough is characteristic and is the most prominent symptom. It is most marked when pigs come out to feed in the morning but can usually be elicited by a period of vigorous exercise. The cough may disappear after some weeks or persist almost indefinitely. The hair coat loses its normal bloom and the skin is grey-tinged. Disturbed respiratory movements are seen only when there are severe lung involvements. Infected pigs do not thrive as well as normal pigs although their appetites are usually maintained. Infected pigs often appear to be progressing satisfactorily unless they are compared to pigs which have not been exposed to the disease. Since all of the pigs in a litter do not suffer equally from the

disease, there may be considerable variation in size of the pigs.

The virus pig pneumonia organism is probably most commonly spread by airborne infection. Piglets frequently become infected from their mothers; on other occasions infection occurs when pigs from several litters are mixed at weaning.<sup>1</sup>

In England, VPP has increased a great deal since the second world war; this is probably due to the rapid expansion of the pig population.<sup>1</sup> The higher concentration of pigs presumably facilitates the transfer of infection from one pig to another.

In the field the incubation period appears to be in the neighborhood of 10 to 16 days. However, a number of pigs infected when young develop an acute pneumonia at 20 to 26 weeks of age. These animals become prostrate, show the conditions of "thumps" and frequently have a rust-colored nasal discharge.

### ***PATHOLOGY***

In swine euthanized on the third or fourth day of infection with swine influenza, the pharynx and larynx are mildly hyperemic and are covered with a tenacious mucus. The exudate is also noted in the trachea and may fill the smaller bronchi and bronchioles. The changes in the lungs are constant and are characteristic of the disease. The involved tissue is a deep purple-red color and is definitely demarcated from the normal tissue. The gross tissue appears as a massive atelectic pneumonia. Lesions are usually limited to the apical, cardiac, and azygous lobes.

The tissues of fatal cases of swine influenza appear somewhat different. There is usually a sero-sanguineous pleural exudate while the bronchial exudate is often blood tinged. The diaphragmatic lobes exhibit a hemorrhagic pulmonary edema while the cardiac, apical, and azygous lobes are consolidated. The predominate cell in the exudate is the polymorphonuclear leukocyte. There are a few lymphocytes in the exudate with a few desquamated epithelial cells. Cervical and mediastinal lymph nodes are swollen and edematous. Ordinarily there are few other pathological changes in uncomplicated swine influenza.

Young and Underdahl (1960)<sup>15</sup> state that typical lesions of virus pig pneumonia consist of plum-colored to reddish brown consolidated areas in the lungs. In the majority of cases the lesions are confined to the ventral portions of the lungs, particularly in the cardiac and apical lobes. Sometimes the lesions are grey and have the appearance of lymphoid tissue. In long standing cases there may be varying degrees of fibrosis. Resolution does not commonly occur and occasionally pleuritis and pericarditis are found. The bronchial and mediastinal lymph nodes are enlarged and edematous.

In acute cases dying from the virus infection alone there may be intense congestion and edema of the lungs. Pigs dying at 20 to 26 weeks of age in herds in which the disease has been present for some time frequently have yellow necrotic areas in the lungs. Pleuritis, pericarditis, and peritonitis are seen in these cases.

Gordon(1963)<sup>3</sup> states that he found more left lungs than right lungs to be normal. This finding may be related to the anatomical differences in the bronchial trees and in the size of the two lungs. It has been noted that the bronchus to the right apical lobe leaves the trachea anterior to its bifurcation and that the total lumen size of the right bronchial tube is greater than that of the left. Correspondingly, the right lung is larger than the left.

It was noted by Scotozza(1963)<sup>8</sup> that pneumonic lesions were present in animals not showing clinical signs as well as in those with typical enzootic pneumonia (VPP). Moreover, pneumonic lesions were more extensive and evident 20 and 30 days after infection than in those observed at 10, 60, 90 and 120 days. Upon histological observation he found an interstitial pneumonia particularly evident in animals killed two months after infection. Hyperplasia of the peribronchial lymphatic tissue characterized by a large number of mitotic figures, some abnormal cells, and invasion of bronchial and peribronchial tissue was found. The hyperplasia appears to be cancerous on occasions. The interstitial process appears less evident in the lungs of animals

killed 10, 20, and 30 days after infection.

Pulmonary lymph nodes showed simple lymphadenitis in pigs killed up to 30 days after infection and in those killed after that period, the lymphadenitis became hyperplastic. So far as the lymph node tissue is concerned, upon inoculation it was able to give rise to clinical signs and lesions of enzootic pneumonia when it was collected 10, 20, 30, and 60 days after infection. Thereafter, (90 to 120 days) it lost such ability but gave rise to a silent infection whose presence appeared only through histological examination of the lungs.

## DIAGNOSIS

The diagnosis of swine influenza as a herd condition is usually not difficult. The history of an outbreak of respiratory disease involving most of the swine in a herd at one time occurring in late fall or early winter is suggestive of swine influenza. Swine influenza may possibly be confused with hog cholera. However, hog cholera begins more insidiously and does not show any respiratory tract involvement that swine influenza does. Observations of the pathological changes at necropsy should definitely settle the diagnosis.

The diagnosis of virus pig pneumonia may be confused with a number of conditions. The coughing and unthrifty condition of an ascarid infection may be confused with VPP. It is believed that many cases diagnosed as ascarid pneumonia are probably actually VPP.

Some think that dry feeding causes pigs to cough, but VPP differs markedly clinically and pathologically from pneumoconiosis. Betts(1952)<sup>1</sup> states that he fed pneumonia free pigs dry feed and saw no coughing.

Swine influenza may be confused with VPP even though swine influenza is a condition of shorter duration and is seen essentially in the autumn and winter. Swine influenza is a more acute and severe disease with pathological lesions different from those of VPP.

Ferkel-grippe or piglet influenza and infectious pneumonia<sup>6</sup> as described in the

literature are probably conditions identical to virus pig pneumonia.

### TREATMENT AND CONTROL

There is no specific therapeutic treatment for swine influenza although careful nursing is very important. Leaving the affected animals alone in a dry non-drafty, warm place is probably the best treatment. Dust aggravates the condition because it irritates the inflamed mucous membranes and increases the coughing. The animals should not be handled and they should have plenty of fresh clean drinking water.

Animals that have recovered from swine influenza are ordinarily immune to clinical reinfection. Young and Underdahl(1955)<sup>14</sup> state that it is well established that injections of either live or formalized influenza virus stimulate immunity to influenza in swine. However, there is no practical need for the procedure so there is not commercial vaccine available at this time.

Influenza is usually seen only in the months of October through January and the virus does not remain in the lung tissue after recovery of the host. The virus lives in the lungworm of swine during the months between the seasonal attacks and is transmitted by this intermediate host. The swine lungworm has an intermediate host, the common earthworm. Therefore, the earthworm is of importance in the transmission of swine influenza. As a rule, swine infected with lungworms carrying the virus not not develop swine influenza immediately as might be expected. Instead, they remain outwardly normal but all that is required to bring forth a severe influenza attack is some stimulus or stressful condition.

Virus pneumonia of pigs is a widespread chronic disease of pigs which is becoming increasingly important as more pigs are raised closely together in large groups in confined areas. There has been much debate on the economic effects of the disease. Therefore, although the disease can be eradicated from a herd, there are many who argue that the gain is small. There are a number of reasons for

the differing views on the economic effects of virus pneumonia of pigs. First, it is difficult to determine the costs of a non-fatal disease. Second, it is not obvious how localized pneumonic lesions depress the average conversion ratio of growing pigs to the extent that has been claimed by Goodwin (1963)<sup>2</sup>. Third, the effects of the disease vary greatly between different herds as well as between individuals of a single herd. One of the most striking differences noted in pneumonia free herds is the evenness of the litters as compared to infected herds.

Experimental findings by Betts(1952)<sup>1</sup> support the field experience which suggests that the infection persists in the pig from one generation to the next and once the disease is introduced into a herd it tends to remain indefinitely in an endemic form. If the animals are kept warm, have dry, draft free sleeping quarters and are adequately fed, the effect of the disease is lessened considerably.

### SUMMARY

The etiology of viral respiratory disease in swine is a confusing issue. Most authorities now believe that respiratory disease in swine caused by viruses is either swine influenza or virus pig pneumonia which is also called enzootic pneumonia, especially in England. Infectious pneumonia, Fergel-grippe, and piglet influenza which were all described to be distinct clinical entities at one time are now all considered to be the same disease condition as virus pig pneumonia.

Swine influenza, which is a seasonal disease showing severe respiratory symptoms for a short period of time, is caused by the swine influenza virus and the bacterium *Hemophilus suis* acting in conjunction. Virus pig pneumonia is caused by a single virus and is a chronic disease that shows only moderate clinical signs. Virus pig pneumonia is very widely spread and costs hog producers millions of dollars annually due to the poorer feed efficiency and the slower rate of growth seen in the infected pigs. There are enzootics of swine influenza every fall but this disease is not nearly as widely spread

as VPP and it is not the economic problem to the hog producer that VPP appears to be.

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#### Kaopectate—R. L. Grier

Kaopectate (Upjohn Co.) is effective in the treatment of acute infectious diarrheal disease. Both kaolin and pectin adsorb toxic products of bacterial growth as soon as contact is made. Such symptomatic treatment of diarrheas is often as important as killing the infectious organisms. Commercial products are available with various antibiotics added to kaopectate. The major side effect stems from prolonged usage, wherein vitamin deficiency arises because adsorption of vitamins has been interfered with by the medication. Effective doses are: horses, cattle—6-10 oz., every 2-3 hours; colts, calves—3-4 oz. every 2-3 hours; and dogs, cats—1-3 tablespoonfuls every 1-3 hours. Administration is oral.

#### Centrally Acting Emetics—Wayne Fawver

Apomorphine is effective by IV, Sub-Q, IM and conjunctival sac injection. The longest latency is after Sub-Q injection, with emesis occurring in about 20 minutes. Copper sulphate given orally induces emesis by stimulation of peripheral receptors, but given IV it stimulates the emetic chemoreceptor trigger zone. The

dose of copper sulphate for dogs is 3.2 mg/kg, IV.

#### Effects of Vegetable Astringents on the Digestive Tract—G. Van Gelder

Vegetable astringents usually contain tannic acid and are employed to alleviate diarrhea. They act by forming protein precipitates which form a barrier and coating over the mucosa. The tannic acid is primarily liberated in the upper intestine and most of the action is exerted there. Dosages have not been carefully studied, nor were there any comparative effectiveness studies reported.

#### Effects of Atropine on the Intestinal Tract—H. T. Holcomb

As with all parasympatholytics, atropine tends to be a spasmolytic and inhibitor of secretions. The most common dosages used range from 0.1 to 0.2 mg/kg; however, some workers recommend even higher doses (2-6 mg for dogs). A subcutaneous dose becomes effective in about 30 minutes. Motility is inhibited best in the distal colon. The drug also decreases gastric secretions and increases the uptake of water and salt from the jejunum. No comparative data were provided.